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During the recent pandemic of influenza I had an opportunity to study the laryngeal and tracheobronchial pictures in influenza patients in hospital and private practice. Some of them were supposed to be suffering from diphtheritic laryngeal stenosis, until direct laryngoscopy and bronchoscopy revealed the true nature of their respiratory difficulty.

For some time it has been my routine to make direct laryngeal and bronchoscopic examinations on patients suffering from croup admitted to the Willard Parker Hospital to ascertain the cause of the stenosis. In a number of cases examined during the past year, we were surprised to see that there was no diphtheritic membrane present. At times the larynx was fiery red, and the ventricular bands and vocal cords were edematous and stippled with whitish points on a deep red background. In these cases the edema was supraglottic and interfered with inspiration. In other cases the voice was not lost as in the supraglottic type and the larynx was apparently normal though the obstruction to respiration was extreme. These cases showed the subglottic and tracheal regions to be the chief site of the lesion. In some instances the lesion was confined to the lower trachea and bronchi, and accompanied by symptoms of asthma. In other cases the entire airway was involved.

Apparently, there are four distinct types of respiratory involvement according to the location of the lesion: (a) Laryngeal; (b) tracheobronchial; (c) bronchopulmonary or asthmatic, and (d) pneumonic.

A. The Laryngeal Type.—This type of influenzal croup closely resembles diphtheritic croup, and is indistinguishable clinically unless a direct laryngeal examination is made. Sometimes a feature of influenzal croup is the absence of gradual progressive rhythmic inspiratory and expiratory dyspnea which is frequently present in diphtheritic croup. At times in influenzal croup there is a persistent recurrent glottic spasm which is very distressing to the patient. This may produce a well marked inspiratory crowing sound, and if subglottic stenosis be present there is an expiratory croupy cough. The cough may be the combination of a whoop and a croup. In influenza the

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larynx and trachea are extremely irritable. The recurrent whoop and paroxysmal cough may even suggest pertussis. The whoop is caused by glottic spasm together with tumefaction of the ventricular bands which tend to close the glottic aperture. These children may become very blue and require intubation. Croup is purely an expiratory phenomenon, and is due to edema and inflammation of the subglottic and tracheal regions. When the stenosis is marked physical examination of the chest may show that no air enters the lungs. Diminished respiratory murmur or a unilateral bronchial obstruction cannot be elicited until after the airway has been made free by the introduction of an intubation tube or bronchoscope.

B. The Tracheobronchial Type.—In these cases there may be a marked tracheobronchitis for several days before definite signs of respiratory difficulty or a croupy cough develops. In these cases the voice may be normal and the child may cry aloud though dangerous obstructive symptoms are present. Intubation may be necessary to save life, because of extensive subglottic and tracheal edema. The larynx usually appears normal, but when a tracheoscope or bronchoscope is introduced into the trachea, the latter is seen to be fiery red and edematous. When the edematous flaccid posterior tracheal wall comes in contact with the anterior edematous mucosa the tracheal lumen and expulsive cough may be shut off almost completely. The trachea and bronchi may present a stippled appearance with scattered adherent plaques of tenacious mucus which may be blood streaked. Collections of gluelike mucus may completely obstruct a bronchus, and in the moderately severe types aspiration of the tenacious secretion may induce recovery if there be no lung involvement.

C. The Bronchopulmonary or Asthmatic Type.—In these cases the onset may be extremely violent or the condition may be ushered in with all the premonitory symptoms of laryngotracheobronchitis. The child may be stricken with a fulminating influenzal asthma even while asleep. This may be mistaken for some form of food anaphylaxis, and the history may bear this out if we are told that the child went to bed in good health aside from a slight cold in the head. The lungs are enormously engorged with blood, and there is extreme difficulty with respiration. Violent glottic spasm may be present and add to the gravity of the picture. The cough may be lost completely. These patients rasp and wheeze and are profoundly prostrated. There is a short stridulous inspiration and a prolonged wheezing expiration audible at some distance. On introducing the bronchoscope we may be surprised to find very congested bronchi comparatively free from secretion. A few streaks of tenacious bloody mucus may be present. As the secretion is poured out the pulmonary congestion decreases.

Many of these children succumb within a few hours after the onset of the attack. When secretion is profuse it may be aspirated bronchoscopically and afford relief. Dr. Jesse G. M. Bullowa agrees with me in the belief that marked spasm of the bronchi is not the cause of the asthmatic attack. Spasm is not seen bronchoscopically. That the cause is probably due to the enormous engorgement of the lung producing inelasticity. The markedly engorged inelastic lung is not expanded on inspiration so that the functionating elastic vesicles are overdistended, producing acute pulmonary emphysema. If the condition becomes extreme, interstitial or tissue emphysema may result. In this type of case the absence of cough may be noteworthy, even on the introduction of the bronchoscope. These children may be able to cry aloud at times. Their difficulty is expiratory. Epinephrin hypodermically may afford relief. Finally, (d) there may be the lobar or bronchopneumonic types of dyspnea, with characteristic air hunger.

The following cases are some of those studied during the epidemic.

REPORT OF CASES

Case 1.—A girl, 4 years of age, had a persistent croupy cough which came on four days after an ordinary catarrhal cold and slight bronchitis. The croupy cough continued to grow worse the following day and a dose of antitoxin was administered. As the croup did not improve within the next twenty-four hours, and as a marked glottic spasm was an added complication, the late Dr. Ralph Whitcher asked me to determine the cause of the laryngeal obstruction by direct observation. The larynx was slightly reddened. The aryepiglottic folds and ventricular bands were stippled. The cords were red. There was a moderate degree of subglottic edema (Plate I, Fig. 3). On the introduction of the direct speculum the glottic spasm was greatly increased. A 5 mm. bronchoscope was promptly introduced which opened the glottic aperture and gave immediate relief to inspiration. The tube, however, met with slight resistance in the subglottic region which was edematous. The tracheobronchial lesion was studied for three minutes. The trachea was red and stippled like the larynx, there was little bronchial secretion (Plate 2, Fig. 3). There was no evidence of diphtheritic membrane. Suction cultures aspirated into a sterile bottle were reported by Miss Valentine as showing pneumococci and a few short rods resembling influenza bacilli. No diphtheria bacilli were present.

After the removal of the bronchoscope the child breathed easily, and the slight amount of dilatation of the subglottic region greatly improved the croupy cough. The following day the child was greatly improved, and aside from an occasional mild whoop she seemed on the road to recovery. The expiratory croupy cough had practically disappeared. The case was one of laryngotracheobronchitis without pulmonary involvement.

Case 2.—Male infant, 11 months old, was apparently ill with an ordinary cold on the chest. There were a few scattered râles over both lungs. His four years old sister had suffered the week before with croup and had received diphtheria antitoxin though the cultures from the nose and throat were negative. Immunizing doses of antitoxin were given at that time to the other children in the family. A few days later the baby became very dyspneic and was given at this time a curative dose of antitoxin. Three days later the croupy symptoms became extreme, and it was at this time that I was called by Dr. Whitcher to make a direct laryngeal examination, for he suspected influenzal

laryngeal edema as being the probable cause of the difficulty on account of the absence of response to antitoxin.

The physical signs in the chest at this time revealed a diffuse bronchitis though very little air was entering the lungs. The temperature was 102.4 F., the pulse was uncountable, respiration was labored. A rapid direct examination showed only a slight reddened larynx. The vocal cords were apparently normal (Plate 1, Fig. 4). The dyspnea was urgent and the cyanosis marked. A 4 mm. bronchoscope was introduced immediately between the apparently normal cords. The subglottic and tracheal regions were very edematous and the tube was introduced through a ring of annular edema with considerable difficulty. There was no evidence of diphtheritic membrane. The trachea and both main bronchi were very red and there were many scattered patches of mucopus on the walls, which were aspirated into a sterile bottle for examination. Miss Valentine reported streptococci in abundance, pneumococci and influenza bacilli.

The infant was able to breathe comfortably through the bronchoscope. Five minutes after its removal intubation became necessary. A small flat headed bulbous noncough-up tube was introduced. Following the examination the temperature rose to 103 F., but no definite signs of pneumonia could be elicited.

There was a marked tracheobronchitis and many râles produced by the rattle of the mucus in the intubation tube. On the fourth day after intubation the temperature fell to 100 F. and the tracheobronchitis was greatly improved. The intubation tube was removed on this day but within an hour, because of subglottic edema, it was replaced. Because of the reaction, cough and increased tracheobronchitis no further attempt was made to remove the tube for six days. On the second trial the persistent subglottic edematous stenosis necessitated the reintroduction of the tube within thirty minutes. Glottic spasm also added to the discomfort during this short period of detubation. The general condition of the child gradually returned to normal three days after the second detubatory trial. We then decided to leave the tube in for one week before the next trial removal. This time on removing the tube the 4 mm. bronchoscope was immediately introduced with ease. It was left in for a few minutes and then gradually withdrawn. The infant was able to breathe fairly well after the removal of the bronchoscope, and made a complete recovery.

Case 3.—A boy, 10 years old, had been in perfect health until he became overheated by skating. That night he was seized with violent dyspnea and asthmatic breathing. Dr. Isadore Friesner recognized the existence of an obstruction in the larynx or trachea below the cords, because the boy was able to speak with a normal voice when the respiratory difficulty temporarily improved. Dr. Friesner desired a direct laryngeal and bronchoscopic examination to determine the cause of the obstruction.

By physical examination of the chest it was found that much less air entered the right lung than the left. These findings were confirmed by Dr. Koplik. There was slight cyanosis.

On direct examination the larynx was slightly cyanotic but otherwise normal in appearance. The vocal cords and ventricular bands were not swollen. There was marked subglottic edema which looked dark and grayish (Plate 1, Fig. 1). A mass of tenacious mucus clung to the interarytenoid space. The 5 mm. bronchoscope was introduced with moderate difficulty on account of the firm subglottic edema. The urgent dyspnea immediately disappeared, but a slight asthmatic wheeze persisted though the bronchoscope had reached the carina of the trachea. There was no evidence of diphtheritic membrane. The trachea had a purplish red color, and there was a thick glue-like exudate covering the posterior wall. The carina was somewhat dusky with a thick tenacious blood stained plaque of mucus on the posterior wall (Plate 2, Fig. 1). This was aspirated into a sterile bottle and the right bronchus was explored. The right bronchus was almost completely filled with thick tenacious bloody

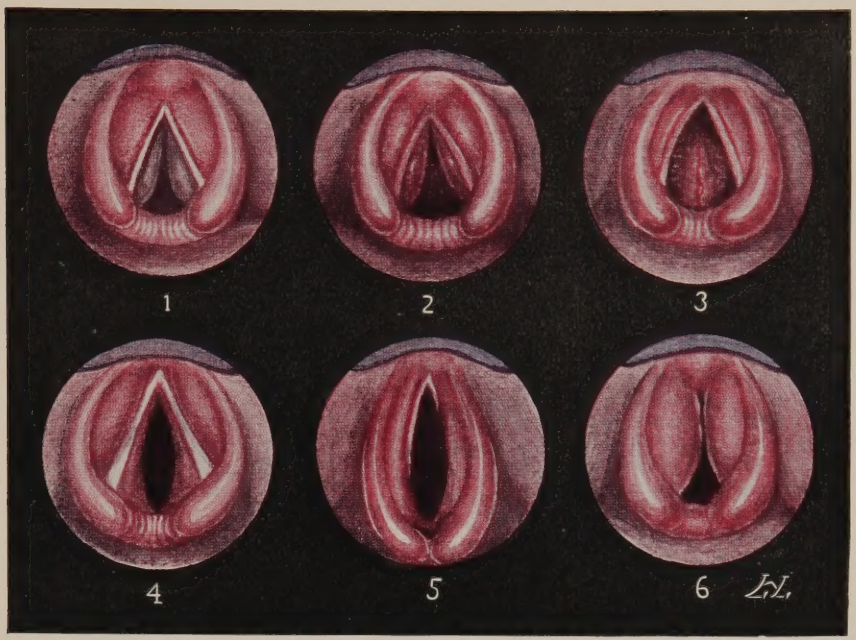

PLATE I

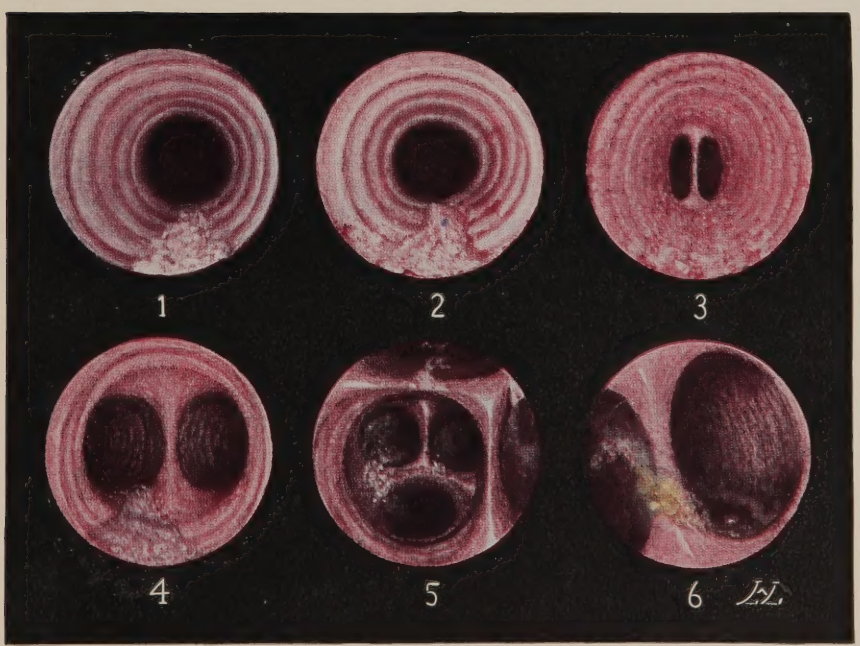

PLATE II

mucopus, which constantly poured from the superior, middle and inferior branches (Plate 2, Fig. 5). The left main bronchus and its branches were comparatively free from secretion, and there was little sign of bronchial inflammation. All of the secretion was removed by suction and the boy's condition greatly improved.

The examination without anesthesia lasted for five minutes. The cough reflex was not lost. There was a gradual return of the dyspnea, and four hours later a second bronchoscopy was performed and secretion removed with immediate relief. An intubation tube was then introduced to prevent recurrence of subglottic stenosis and in a short time the boy fell asleep.

The following day the general condition became worse and we again bronchoscoped him. There was little or no improvement though the secretion was aspirated. The right main bronchus was beefy red and edematous. The temperature was 104.3 F., respirations 52 and pulse 100. The marked cyanosis was only slightly relieved by bronchoscopy and intubation. The cough reflex was lost, and the boy was unable to expel the rapidly forming secretion. He sank rapidly on the third day of his illness. He was delirious, cyanosed and unable to cough. The respirations were labored and the lungs were filled with bubbling râles. The urine was bloody and scant. Twenty-four hours later he succumbed to massive pulmonary involvement. The suction cultures taken from the tracheobronchial tree showed *Streptococcus hemolyticus* in abundance.

Case 4.—A boy, 3 years of age, ill three days, was admitted to the Willard Parker Hospital suffering from laryngeal diphtheria. The child was very croupy and a direct laryngeal examination disclosed a membrane on the ventricular bands and vocal cords. Dr. Thompson intubated the patient shortly afterward. A dose of 10,000 units of diphtheria antitoxin was given intravenously. Direct cultures, taken just before intubation, showed diphtheria bacilli. The intubation tube was worn for four days and then removed. The boy remained without the tube, and after having had a series of negative cultures from the nose and throat was ordered for discharge.

On the day set, the temperature suddenly rose to 104.8 F., and there was a rapid return of the croup, for which condition he had been admitted. It was first thought that the recurrent croup was due to subglottic stenosis following diphtheria. However, the fluctuating temperature and diffuse bronchitis suggested influenza, especially in the presence of the epidemic and the numerous cases in the hospital.

A direct examination revealed a beefy red larynx with marked tumefaction of the ventricular bands (Plate 1, Fig. 6). The subglottic region was red and edematous though the 5 mm. bronchoscope was introduced without much difficulty. The trachea and bronchi were reddened and there was a moderate amount of bronchial secretion coming out of the left bronchus (Plate 2, Fig. 4). Suction and swab cultures taken from the larynx, trachea and bronchi showed no diphtheria bacilli, but pneumococci and influenza.

The bronchitis and fluctuating temperature lasted until the end of the fifth week. Reintubation was not necessary after the removal of the bronchoscope, for it had dilated the subglottic area sufficiently to afford relief. This case illustrates the sequence of two different infections each causing acute laryngeal stenosis. The child recovered.

Case 5.—A girl, 6 years of age, was admitted to the Willard Parker Hospital after intubation in her home by the ambulance surgeon. On admission the child was in poor condition. The temperature was 105.2 F., pulse uncountable and respirations 58. She had received 10,000 units of diphtheria antitoxin at the outset of the croup two days previously. Little air entered the lungs in spite of the tube, but because of the desperate condition of restlessness and delirium, it was deemed wise not to disturb her. She seemed moribund. The lungs were filled with moist râles, she was deeply cyanosed like all dying influenza patients.

We were surprised the following day to find the patient still alive and apparently improved. Epinephrin had been administered, but the dyspnea and cyanosis were not completely relieved. By direct laryngoscopy the tube was removed. The pharynx was red with granular white spots on the postpharyngeal wall, and also on the buccal mucosa. There was no diphtheritic membrane present. The larynx was beefy red and stippled with small white points. The trachea was swollen, but there were no apparent hemorrhages. The distended vessels were plainly visible. Thick ropy blood streaked secretion partially obstructed the lower trachea and bronchi. There was no cough on the introduction of the bronchoscope even when the bifurcation of the trachea was reached. There was slight asthma present with the bronchoscope in place. The child was delirious during the manipulation. After aspiration of secretion the extreme cyanosis was slightly relieved. The intubation tube was reintroduced. Cultures taken by suction showed no diphtheria bacilli, pneumococci and a few influenza bacilli.

On the third day after admission the temperature rapidly fell to 100 F., pulse and respiration were unchanged. Five days after admission, the child was still wearing the tube and still delirious. Areas of bronchopneumonia were found in the chest on physical examination. There was slight cyanosis. On the seventh day the tube was removed but replaced in five minutes because of subglottic edema. On the eleventh day after admission the temperature was normal and the child's general condition was good. Dr. Thompson removed the tube and the child remained without it for six days, when she again became dyspneic. The area of pneumonia seemed more extensive on physical examination and the temperature rose to 103 F. Two days later the child was again intubated to relieve a distressing spasm of the glottis and the retention of secretion in the lung. The following day the child coughed up the tube and remained without it thereafter. After a convalescence lasting five weeks she was discharged cured.

Case 6.—An infant, 11 months of age, was seen in consultation with Dr. Sheitlis, and intubated shortly afterward. The baby had had a slight cold in her head and after a few days in bed was pronounced well. A few days later the coryza recurred, the temperature rose to 103 F., and there was a slight cough. After several days the bronchitis subsided. Two days later the baby became slightly croupy at night. Diphtheria was suspected and a dose of 10,000 units of antitoxin was administered. Cultures taken from the pharynx showed abundant pneumococci and influenza bacilli, but no diphtheria.

The following day the spasm of the glottis was extremely bothersome, and there was an inspiratory crow and an expiratory croupy cough. Ipecac relaxed the spasm for an hour. After violent spasm she became cyanosed. The antitoxin had no effect and after three days the baby was decidedly worse. The patient was very limp and pale. She had just finished a violent paroxysm of coughing. Within twenty minutes there was a recurrence of the attack. There was a marked inspiratory crow on each respiration, and a severe croupy or barking expiration. The crying voice was hoarse. Cyanosis was marked. Gradually the attacks subsided.

On direct inspection of the larynx the mucous membrane was of a dull grayish red color. There were small stippled patches on the ventricular bands which were much swollen. The vocal cords were only partly visible on account of the constant spasm and were red (Plate 1, Figs. 2 and 5). The subglottic region was very edematous so that the 4 mm. bronchoscope was introduced with difficulty. In this locality there were small patches of sticky mucus. The child made no attempt to cough until the trachea and bronchi were freed of the thick gluelike secretion by suction (Plate 2, Fig. 2). Both inspiration and expiration improved. A flat headed noncough-up tube for a 1-year-old child was introduced after the removal of the bronchoscope. The baby's condition gradually improved and after three hours she was comparatively comfortable, though the spasmodic cough, with the tube in place, was troublesome at times.

A few days later the temperature, which had been 103 F., gradually fell to normal and the tracheobronchitis subsided.

Four days later the intubation tube was removed through my direct spatula. The subglottic edema had not subsided, and within thirty minutes reintubation was necessary. Six days later the tube was again removed as the arytenoids had resumed a normal appearance. As the intubation tube was removed the bronchoscope was introduced and secretion aspirated and the condition of the trachea and bronchi observed. On the gradual withdrawal of the bronchoscope the baby was able to breathe through a fairly normal larynx. The baby had a few attacks of glottic spasm and croup but they were not sufficiently severe to require reintubation. After three weeks the baby had recovered.

Case 7.—A boy, 3½ years of age, was admitted to the Willard Parker Hospital apparently moribund. On admission an immediate direct laryngeal and bronchoscopic examination was made. There was considerable difficulty with respiration; expiration was prolonged and wheezy. The larynx was deep red in color and cyanosed. There was slight subglottic edema. The 5 mm. bronchoscope was introduced to the carina and revealed absolutely dry trachea and bronchi. There was dusky redness of the bronchi and trachea. There was absolutely no sign of a cough reflex.

Three hours later the child became so much worse and the asthmatic expiration became so bothersome that the bronchoscope was again introduced but without result. This time there was a slight amount of yellowish tenacious mucopus exuding from the left superior lobe bronchus (Plate 2, Fig. 6). All the other bronchi remained dry. Suction cultures showed pneumococci; no diphtheria bacilli were found. Twelve hours after admission the child died.

EXPLANATION OF PLATES

Plate 1

Fig. 1.—Direct laryngeal view in Case 3. The larynx was slightly cyanotic but otherwise normal in appearance. The vocal cords were normal and the ventricular bands slightly swollen. There was marked subglottic edema. This is shown in the picture as two dark grayish masses projecting from the lateral subglottic walls and well below the vocal cords.

Fig. 2.—Direct laryngeal view in Case 6. The larynx is deeply congested. The vocal cords are reddened and the ventricular bands swollen. The subglottic region is beefy red and swollen and there is a thin exudate in stippled patches on the tumescent mucosa. The arytenoid cartilages are slightly swollen. There was a constant glottic spasm which added to the gravity of the case. The subglottic edema is productive in causing the expiratory croupy bark.

Fig. 3.—Direct laryngeal view in Case 1. The aryepiglottic folds and ventricular bands are swollen. There is a stippled appearance of the larynx and many fine white points are seen on the deep red background. There is moderate subglottic edema present. The markedly injected vessels and white stippled points are plainly seen in the trachea.

Fig 4.—Direct laryngeal view in Case 2. The ventricular bands are slightly swollen, causing inspiratory difficulty. Note the marked annular subglottic and tracheal edematous stenosis which made the introduction of the infant bronchoscope difficult. The vocal cords are normal, but partially covered by the swollen ventricular bands.

Fig. 5.—Direct laryngeal view in Case 6. Showing persistent glottic spasm and the cause of the bothersome inspiratory crow. The swollen ventricular bands overlap the cords and cause inspiratory dyspnea.

Fig. 6.—Direct laryngeal view in Case 4. Note the beefy red larynx with enormous tumefaction of the aryepiglottic folds and ventricular bands. Only a small lumen is left for inspiration, for the swollen ventricular bands while in spasm almost close the glottic inlet. On introduction of the bronchoscope and separation of the tumefied masses the subglottic region was found to be equally edematous. This case had both inspiratory crow and expiratory croup.

Plate 2

Fig. 1.—Tracheal view in Case 3. Note the slightly cyanotic trachea with thick tenacious glue-like secretion of influenza on the posterior wall. The trachea was edematous and there was an asthmatic wheeze even after the secretion had been aspirated.

Fig. 2.—Tracheal view in Case 6, showing tenacious secretion on the posterior wall and hemorrhagic patches on the lateral walls and in between the tracheal rings.

Fig. 3.—Tracheal view looking toward the carina trachialis (Case 1). Note the marked stippling and swelling of the mucous membrane. The white points are pneumococcic stippling on the reddened and blood streaked mucous membrane. There was very little secretion removed by aspiration.

Fig. 4.—Tracheal view at the carina in Case 4. There was no involvement of the right bronchus which was free from secretion. There is a plaque of tenacious mucus partially occluding the left main bronchus. Note the hemorrhagic points surrounding the swollen carina.

Fig. 5.—Bronchoscopic view of the right main bronchus and its branches in Case 3. The stem bronchus and branches are very cyanotic and completely occluded with thick bloody mucopus which obstructs respiration on that side entirely. No air entered the right lung until after the secretion was aspirated bronchoscopically. There was a decided asthmatic wheeze on expiration even after the aspiration of secretion and with the bronchoscope in situ.

Fig. 6.—Bronchoscopic picture of the left main bronchus and left superior lobe branch (Case 7). The child was admitted in a moribund condition. No secretion was found in the bronchi on the first bronchoscopic examination and only a small amount was poured out at the time of the second bronchoscopic examination and then only from the left bronchus. The larynx was not involved. The child was suffering from the bronchopulmonary asthmatic type of influenza and had no cough reflex on the introduction of the bronchoscope. Note the tenacious glue-like mucopus exuding from the left superior lobe orifice.